Abstract

Disclosed is a transgenic knockout mouse whose genome comprises a homozygous disruption in its endogenous FcRn gene. The homozygous FcRn disruption prevents the expression of a functional FcRn protein, resulting in a transgenic knockout mouse in which exogenously administered IgG1 exhibits a substantially shorter half-life, as compared to the half-life of exogenously administered IgG1 in a wild-type mouse. The transgenic knockout mouse with a homozygous FcRn disruption is also unable to absorb maternal IgG in the prenatal or neonatal stage of development. Also ~~described~~disclosed is a transgenic knockout mouse comprising a homozygous FcRn disruption and a human FcRn transgene. The transgenic addition of human FcRn results in a substantial increase in the half-life of exogenously administered human IgG1. Methods of using the transgenic knockout mouse, and cells derived from them, are also disclosed.